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# Severe Decompression Illness Following Simulated Rescue from a Pressurised Distressed Submarine

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## Summary

If adequate transfer under pressure or recompression assets were not available after rescue from a pressurised Disabled Submarine, the rescuees may suffer from severe or fatal decompression illness (fDCI). Effective methods of reducing the risk of fDCI require characterisation. This study uses a large animal model (goat) to estimate the dose (pressure) response (fDCI) relationship. It also addresses the putative intervention measures of breathing oxygen after surfacing or slowing the rate of decompression, as much as the operational cycle time of the rescue vehicle will allow.

The efficacy of interventions was determined by exposing a group of twelve animals to the LD<sub>75</sub> pressure. After surfacing at the standard rate, oxygen was delivered by oro-nasal mask for one hour. Alternatively, animals were decompressed through a slow, stepped decompression profile, designed to prevent any microbubble formation on ascent to the surface. Animals were observed for signs of decompression illness (DCI) for up to 10 hours post decompression. They were then humanely killed for necropsy. Animals showing continuously declining vital signs were considered to be dying and were humanely killed.

It was shown that respiratory DCI is the most likely cause of death after rapid decompression from deep air saturation. Interventions, such as slowing the rate of decompression, which reduce the bubble load on the lungs on surfacing are likely to be the most effective. Non-recompression therapies, which target improving gas exchange in the lungs, should also improve the outcome.

## Introduction

The submarine's primary role is to deliver its payload to the selected target. This requires it to be covert, fast and manoeuvrable. Constraints are present which prevent the designer from building a totally reliable boat and despite the emphasis on design for high reliability, equipment failures can and do happen. The Russian submarine KURSK highlights the most recent example of such an incident. The covert nature of the submarine's role also makes it vulnerable to collision when at or near the surface, e.g. the Peruvian boat PACOCHA (Harvey, 1989). In both of the cases cited above, further complications occurred when flooding raised the ambient pressure within the boat, increasing the likelihood of rescuees developing serious decompression illness (DCI) on reaching the surface.

If the pressure is greater than about 1.7 bar for more than 24 hours, the survivors will require a controlled decompression to avoid DCI (Bell *et al*, 1986; Eckenhoff *et al*, 1986). Logistical constraints to operators of submarines with large crews ( $>\sim 80$ ), may mean that rescue vehicles arrive at the scene prior to the arrival of the transfer under pressure facility. If the conditions in the submarine are deteriorating, then the on-scene commander will have to consider commencing the rescue operation without the means to control the decompression or to treat the survivors for DCI.

Limb pain only decompression illness has frequently been shown to respond to delayed therapy (Bennett and Elliott, 1993), with little risk of long term adverse health effects. Experience of diving accidents has shown that severe 'missed' decompression can cause permanent neurological damage or even be fatal. The rescue teams require advice to allow the best decisions to be made under such adverse circumstances.

Obviously these "bad outcomes" need to be avoided if at all possible. There is a lack of information upon the relationship between pressure exposure and risk of a bad outcome, which needs to be elucidated. Additionally, any non-hyperbaric methods of reducing the risk of a bad outcome (i.e. prophylactic measures) for a given pressure exposure would be of great advantage and also warrants thorough investigation. Obvious ethical considerations prevent the execution of this study with human volunteers. Therefore, the US Navy has sponsored a three Centre study using large animal models to provide the best data on which to base the advice to be given to on-scene commanders.

## Methods

The goat has been used in our laboratory for many years, having been shown to be a good model for human decompression illness (Boycott *et al*, 1906; Seddon, 1997). The species has been found to be slightly more resistant than man to DCI arising from long near saturation exposure. Previous work has demonstrated that for practical purposes 24 hours exposure to raised pressure is sufficient to achieve saturation (Seddon, 1997). The protocols used here were approved by our local animal use review board.

### Dose Response Curve

To determine the dose response curve, 48 adult female and male castrated goats were exposed in groups of three to an ambient pressure in the range of 55-85 fsw for 24 hours. Mean body mass was 48 kg, with a range 36 - 62 kg. No animal was exposed to pressure in the preceding 4 weeks to avoid risk of acclimation hyperbaric exposure. If an animal had experienced DCI in a previous study it must have been shown to have fully recovered, following a single hyperbaric treatment on USN Table 5 (RN Table 61), before entering the present study. The animals were exposed to 2.67 – 3.58 bar (55 - 85 fsw) for 24 hours, with compression on air at 1.0 bar/min, inside a 14m<sup>3</sup> chamber. Environmental gases were such that CO<sub>2</sub> <0.2 kPa, O<sub>2</sub> = 20.9+/- 0.2% (v/v) and CH<sub>4</sub>< 0.1 kPa throughout the exposure. The ambient temperature remained at 15 - 22°C, except during pressure moves upon initial compression and decompression to the surface. Food and water were given to the animals *ad libitum* up to 8 h prior to decompression, then food alone was withheld. Decompression following the 24 h exposure occurred at 1.0 bar/min (except in 9 animals at 3.46 bar (82 fsw) where decompression occurred over 15 min; there was no difference in outcome in these cases).

Although goats are hardy, feral animals, they do express discomfort strongly, either through vocalisation or altered body language. A protocol for managing pain was developed in the early stages of the experiment. Intra-venous (IV) Torbugesic was administered within 5 min of surfacing. Subsequently Benzodiazepam was given by slow IV injection to sedate the animals if required. The animals were observed for up to four hours after surfacing. If at 4 hours the animals were showed no signs of terminal cardiorespiratory or CNS damage, it was assumed that they would survive. The following clinical signs were recorded:

- Presence of limb pain
- Motor control
- Respiratory rate, pattern and end tidal gases
- Heart rate
- Arterial oxygen saturation by pulse oximetry
- Cyanosis
- Blood gases (CO<sub>2</sub>, O<sub>2</sub> and pH)

The Kisman – Masurel (KM) method was used to detect intra-vascular gas bubbles.

Trans-thoracic 2-D imaging was also conducted on an opportunity basis. The carotid artery and jugular veins were also observed for the presence of bubbles.

Animals were considered to be “bad outcomes” if their vital signs were poor (respiratory rate > 50 and heart rate > 180) and continuing to decline over a 20 min period. Such signs were occasionally accompanied by strong visceral pain, which could not be relieved. It was assumed that these animals too would be bad outcomes, and were killed humanely. Clear indications of cerebral damage were also assumed to be fatal and the animals were killed humanely. Examples included convulsions or nystagmus.

These observations were made at 15, 30, 60 minutes after surfacing and at 30 minute intervals thereafter up to four hours. The time of any significant changes was also recorded. A gross post mortem was conducted at about 5.5 hours after reaching the surface in this component of the study.

### Intervention methods

Two possible prophylactic DCI interventions were tested; post exposure O<sub>2</sub> breathing and a four hour decompression. The estimated lethal dose 75% (ED<sub>75</sub>) point of 3.35 bar (see Figure 2 for dose response curve) was taken as the standard saturation depth. The animals were compressed in the same way as in the dose response study, and the environmental parameters were also maintained as previously. These animals were either decompressed to the surface at 1 bar/min followed by 1 h O<sub>2</sub> breathing or a staged decompression taking 4 h.

Twelve animals were exposed to this pressure exposure in each test and if the incidence of DCI fell to 33% the intervention would be accepted as effective.

### Post exposure oxygen breathing

The effect of oxygen breathing at the surface was tested. 100% oxygen was administered by oro-nasal mask for one hour after surfacing. A limit of one hour on oxygen post surfacing was set, as the provision of unlimited oxygen to 100 plus rescues by open circuit requires vast quantities of bottled oxygen, and is unlikely to be practicable. Also, the gradient of the dose response curve predicts that only a small reduction of gas load is required to produce significant benefits.

In this set of studies, observations continued on the subjects for up to 10 hours (rather than 4 h) depending on the condition of the goat. Animals were sent for post-mortem the following day.

#### Four hour decompression

A four hour decompression profile was also tested following the 24 h saturation period at 3.35 bar. The objective of the decompression profile was to prevent the formation of gas bubbles that would slow further gas washout. To be sure that bubbles had not formed, the first stop had to be at least two hours duration, which would allow KM Doppler scores to be observed. The aim was for a supersaturation ratio ( $PN_2$  tissue/ $P_{\text{ambient}}$ ) as close as possible to 1, though stop times and depths were refined by common sense to avoid confusing decompression rates. A single tissue compartment model (based on previously obtained saturation data) with a half-life of 106 minutes was used to calculate the profile (see Figure 1).

Again, the animals were watched for a period of up to 10 h post surfacing, then humanely killed and sent for post-mortem the following day.

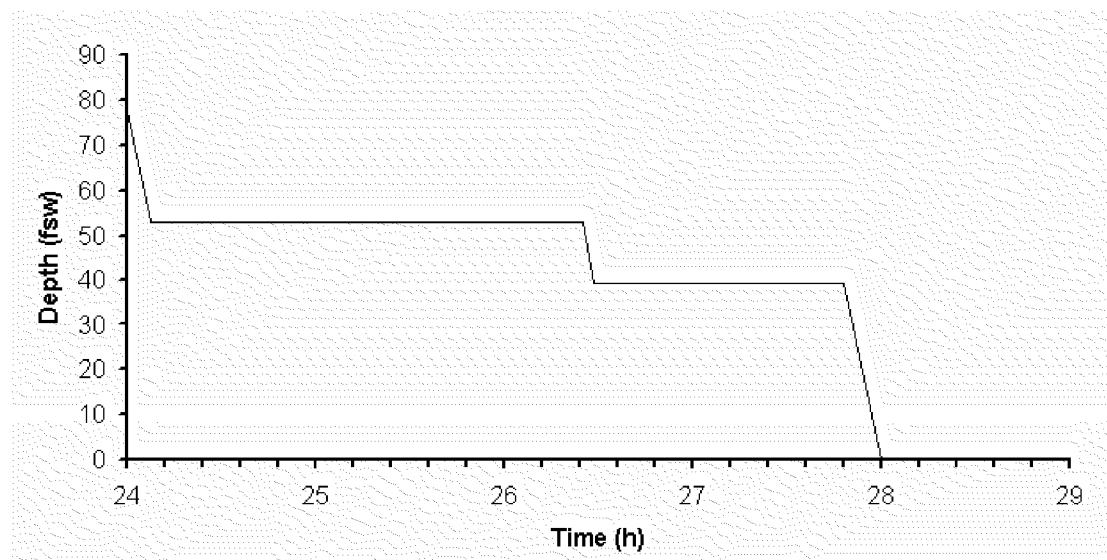


Figure 1 – Slow decompression profile calculated to avoid bubble formation.

## Results

### Dose response curve

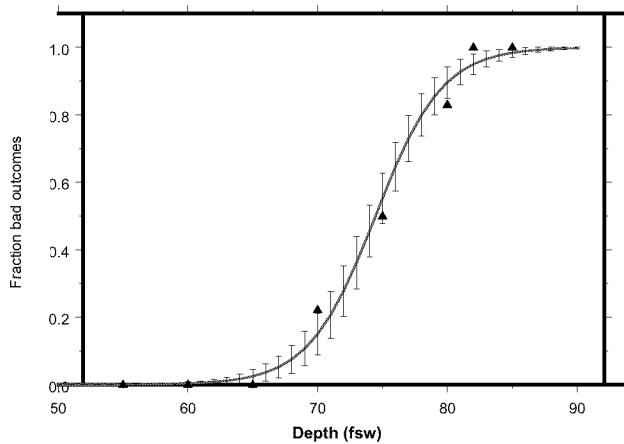
All 48 animals presented with limb pain affecting one or more limbs. All animals also presented with respiratory decompression illness (chokes) to varying degrees. Those least affected had an increased resting respiratory rate, while those most severely affected had severe tachypnea, were hypo-ventilating and were cyanosed. Measurement of venous blood gases showed an elevated  $PCO_2$  and a depressed  $PO_2$ . Three animals presented with central nervous system signs and were diagnosed as bad outcomes. Figure 2 shows the spread of bad outcomes related to depth, producing the dose response curve. From this curve it was estimated that  $ED_{75}$  saturation depth was 3.35 bar (~75 fsw).

Venous gas emboli were present at Kisman-Masurel score of four at all observations. 2D imaging showed relatively few bubbles in the periphery compared with the pulmonary artery. No bubbles were observed in the left ventricle or the carotid artery.

Common examples of post mortem findings are:

- Pulmonary oedema.
- Foam in bronchi.
- Gas in major vessels.
- Haemorrhage in brain/mid-brain/spinal cord (C1 - T5).
- Excess Cerebrospinal Fluid in C1 - T5 area.
- Pale brain coning (compression).

There were no obvious post mortem differentiating signs between the bad outcome group and those that survived.



**Figure 2 - Showing the derived dose response curve for bad outcomes. The error bars are the predicted 95% confidence intervals on the mean.**

#### Post exposure oxygen breathing

In this case, there were five bad outcomes. This was significant at the  $P=0.06$  level indicating a strong trend to a significant benefit. One animal was completely asymptomatic; the 11 remaining animals all exhibited limb bends, while 5 had chokes (choke being defined as increased respiratory and heart rates) and were deemed to be bad outcomes. One of the latter also suffered from severe CNS complications. All animals in this part of the study exhibited KM pre-cordial Doppler ultrasound scores of four at the surface.

The general pathology on post mortem was as in the dose response part of the study.

#### Four hour decompression.

In this trial, the number of bad outcomes dropped to 2 out of 12 (17%,  $P>0.002$  – a significant improvement on the  $ED_{75}$ ). No bubbles were detected during the decompression, fulfilling the aim of the model, however KM scores at surface were still four. Some of the animals were affected by respiratory compromise, but not as severely as the controls. There was only one case of cerebral involvement. Again, the general pathology was the same as the earlier parts of the study.

#### Discussion

It would seem that bad outcomes are largely due to respiratory decompression illness, with a few due to cerebral damage. Therefore, to decrease the risk of a bad outcome, the insult to the lungs needs to be reduced. Oxygen administered post decompression showed a beneficial trend. In addition, anecdotal evidence indicates that oxygen post decompression is effective at reducing the incidence of symptoms following diving accidents. Oxygen administered during decompression should be of even greater benefit as is found in diving and acute altitude exposure. However, at the onset of this study none or very few rescue submersibles are equipped with an oxygen delivery system.

It is accepted that slowing the decompression will be effective, but what is practical? Assuming that the commander wished to evacuate the DISSUB quickly, the Deep Submergence Rescue Vehicle operators have advised that up to four hours could be required to recycle the submersible for its next flight. This would make a four hour stepped decompression to avoid DCI an eminently suitable method of treatment.

Fatalities occurred in the goat model at pressures greater than 2.8 bar saturation. The predicted  $ED_{50}$  is 3.25 bar with 95% incidence at 3.5 bar. Other work is in progress to establish the likely figures for man. This work has shown that slowing the decompression will reduce the risk; oxygen breathing is showing a trend to reducing the risk and it can be inferred that measures to improve gas exchange could also improve the outcome. As respiratory DCI is complicated by

pulmonary oedema, a diuretic may be beneficial as a prophylactic measure, and this option should also be investigated in a further study. All of these three options address different mechanisms; they accelerate gas washout, decrease bubble formation and reduce pulmonary oedema, and therefore combinations of the above may be more effective than any alone. Future work will address this question.

In summary this study has confirmed that severe missed decompression can be fatal but the risk of fatalities may be reduced without immediate need for recompression facilities. Although acute symptoms of missed decompression may be alleviated by the varying intervention methods discussed, the gross pathology of all groups showed a similar level of decompression insult. This indicates that post intervention, rapid hyperbaric therapy should be carried out as soon as practicable to reduce the likelihood of chronic DCI.

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